Serum Lipids and Restenosis After Successful Percutaneous Transluminal Coronary Angioplasty

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The effects of plasma lipids on the clinical and angiographic parameters of 134 patients, in whom coronary angioplasty was performed in 157 vessels, were prospectively examined. During a 6-month follow-up, restenosis was detected angiographically in 39 patients (29%; 45 vessels). None of the clinical, biochemical, or angiographic variables examined was predictive of stenosis and the tendency of a vessel to restenose was not patient-dependent but rather lesion-related. However, restenosis developed in 31 of 102 vessels (30%) in patients with high-density lipoprotein (HDL) cholesterol ≤40 mg/dl, compared with restenosis in 10 of 55 vessels (19%) in patients with HDL cholesterol >40 mg/dl (p = 0.092). No significant differences were observed when restenosis rates were compared in patients with total cholesterol levels >250 mg/dl or <250 mg/dl; no differences were seen in low-density lipoprotein (LDL) cholesterol levels when comparing patients with >160 mg/dl and <160 mg/dl. In 117 patients (132 vessels), complete serial blood specimens were obtained until the concluding angiography at 6 months. During follow-up, both groups (those with and without restenosis) had almost similar findings. Triglycerides decreased equally in both groups, and total cholesterol increased mildly in those who had restenosis; HDL and LDL cholesterol levels increased significantly in each group. No significant differences were observed with respect to extent of these changes between the groups. Thus, although lipid levels at the time of angioplasty and at 6 months follow-up were not found to predict the occurrence of restenosis, the association of low high-density lipoprotein levels and the tendency for restenosis should not be overlooked.

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Restenosis after successful percutaneous transluminal coronary angioplasty (PTCA) occurs in 30% to 40% of patients (range 20% to 70%) within 3 to 6 months[1–7] despite extensive efforts to reduce its incidence with drugs and lipid-lowering regimens.[2–4,8–12] Restenosis may be considered as a multifactorial process of the arterial endothelium resulting from balloon injury and involving mechanical and hemostatic factors and cellular proliferation.[9] If elevated serum lipids adversely affect the healing of endothelium, it could be that an improved serum lipid profile will promote endothelial healing, thus reducing the restenosis rate. Whereas multiple studies have examined clinical and angiographic risk factors for restenosis after PTCA,[2–4,13–15] there is conflicting evidence supporting serum lipids as a predictor of restenosis,[2,3,10] and no consensus on this issue has yet emerged. The present study examines the effects of prospectively measured plasma lipids on clinical and angiographic parameters of patients undergoing PTCA.

METHODS

Study population: This study included 148 consecutive patients, with either stable or unstable anginal syndrome, who underwent successful PTCA without any complications in the subsequent 24 hours. The experimental protocol was approved by the Human Subjects Review Committee of the Tel-Aviv Medical Center and all patients gave written informed consent.

Angioplasty procedures and quantitative analysis: PTCA was performed using standard available balloon catheters, sized to the normal lumen diameter of the artery to be dilated. Inflation duration and pressure were left to the discretion of the angioplasty operator. On completion of the procedure, 10 minutes were allowed to pass before resuming angiograms of the dilated vessel. A final angiogram was performed after 6 months unless the patient's clinical condition necessitated earlier intervention. If no restenosis was present and the follow-up time was within 2 months, the patient was requested to undergo further coronary angiography at 6 months.

Qualitative analysis of coronary morphology was obtained by consensus at a blinded review of the angiograms by a panel of 3 experienced cardiologists. Quantitative analysis of the lesions before and after PTCA and at follow-up was performed, in identical projections, by means of manual edge-detection and computer-assisted calculations as previously described.[16] Successful PTCA was defined as ≥20% reduction in luminal diameter of the dilated segment and <50% diameter stenosis immediately after PTCA. Restenosis was defined as a narrowing of >50% at the site of previous dilation measured from the same angiographic view.

TABLE I Baseline Characteristics of Patients With and Without Restenosis

Variable	No Restenosis (n = 95)	Restenosis (n = 39)
Age (years)	56 ± 10	54 ± 10
Men (%)	75 (79)	35 (89)
Body mass index (kg/m^2)	25 ± 4	26 ± 3
Current myocardial infarction (%)	53 (56)	25 (63)
Previous myocardial infarction (%)	23 (24)	13 (34)
Systemic hypertension (%)	19 (20)	9 (22)
Current smokers (%)	63 (66)	24 (61)
Diabetes mellitus (%)	12 (15)	5 (14)
Hyperlipidemia (%)	31 (33)	11 (28)
Recent thrombolytic treatment (%)	31 (33)	12 (31)
Left ventricular ejection fraction (%)	50 ± 8	48 ± 10
Triglycerides (mg/dl)	175 ± 77	172 ± 64
Cholesterol (mg/dl)	207 ± 41	202 ± 43
HDL cholesterol (mg/dl)	39 ± 9	37 ± 8
LDL cholesterol (mg/dl)	134 ± 29	132 ± 31
Previous treatment (%) with		
Nitrates	67	71
β blockers	54	53
Calcium blockers	48	51
Aspirin	80	81
Heparin	12	11
Lipid lowering drugs (%)	9	9

HDL = high density lipoprotein; LDL = low density lipoprotein.

TABLE II Comparison of Lesion Morphology and Angioplastic Procedure Parameters of Patients With and Without Restenosis

Variable	No Restenosis (n = 95)	Restenosis (n = 39)	p Value
Lesion morphology			
Number of lesions	112	45	
Length (mm)	9 ± 4	9 ± 3	NS
Eccentric lesion (%)	76	73	NS
Filling defect (%)	21	22	NS
Ectasia (%)	18	18	NS
Angioplasty parameters			
Extent of CAD (%)			
1 vessel	59	56	NS
2 vessel	35	31	NS
3 vessel	6	11	NS
Vessels involved (%)			
Left anterior descending	50	55	NS
Circumflex	19	23	NS
Right coronary artery	31	22	NS
Site (%)			
Proximal	22	32	NS
Mid	62	58	NS
Distal	16	10	NS
Left ventricular EF (%)	63 ± 10	62 ± 10	NS
Visible collaterals (%)	16	15	NS
Inflations (n)	4 ± 2	4 ± 1	NS
Maximal inflation P (atm)	8 ± 2	8 ± 2	NS
Maximal inflation D (sec)	159 ± 125	158 ± 99	NS
Balloon/vessel caliber	1.0 ± 0.2	1.0 ± 0.2	NS
% stenosis before PTCA	65 ± 9	68 ± 8	0.054
% stenosis after PTCA	24 ± 12	24 ± 12	NS
Dissection (%)	14	15	NS

D = duration (in seconds); EF = ejection fraction; P = pressure (in atmospheres); PTCA = percutaneous transluminal coronary angioplasty.

Patient management and follow-up: After PTCA, patients were transferred to the cardiology ward where they were monitored and frequent vital signs taken for at least 12 to 24 hours. A continuous heparin infusion (begun during PTCA) was administered for 12 to 18 hours and routinely discontinued 4 hours before the intravascular sheath was removed. All patients were discharged with instructions to take aspirin 125 mg/day to 250 mg/day and diltiazem 90 mg/day to 180 mg/day 1 to 2 days after PTCA; a 12-lead electrocardiogram was recorded at discharge. Patients were seen at the outpatient clinic at 1, 2, 3, and 6 months after PTCA for an interview, physical examination, and a 12-lead electrocardiogram.

Laboratory tests: Overnight fasting blood specimens were obtained at baseline and at follow-up clinic visits, and included determination and measurement of total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides (measured with a Boehringer-Hitachi 737 analyzer, Mannheim, Germany). In addition, complete blood chemistry (Beckman Synchron CX3, Beckman Instruments Inc, La Brea, California), complete blood count, prothrombin time and partial thromboplastin activity, and urinalysis were measured.

Left ventricular function: Patients underwent 2 radionuclide studies performed 24 to 72 hours after PTCA and immediately before the follow-up angiogram. Left ventricular ejection fraction was determined and all radionuclide examinations were assessed by the same cardiologists unaware of the clinical course of the patients.

Data review: The following variables were analyzed: CLINICAL: age, gender, current smoking, diabetes mellitus, hypertension, hyperlipidemia, duration of angina, type of angina or recent myocardial infarction, location of infarction, recent (up to 30 days) use of an intravenous thrombolytic agent, and clinical status during follow-up and upon completion of the study.

ANGIOGRAPHIC: The pre- and postangioplasty features as well as the follow-up features of the dilated artery included the following variables assessed at an end-diastolic frame: dilated artery, proximal mid- or distal stenosis location, 1-, 2-, or 3-vessel disease using the 50% diameter stenosis definition; length of the stenosis; location of the stenosis at a bend of >45°; and branch point location, the preangioplasty presence of contrast staining, or a filling defect suggestive of thrombus and collateral circulation to the dilated artery.

Statistical analysis: All results or continuous variables are expressed as mean ± 1 SD. Differences between continuous variables were compared using the Student's *t* test for 2 groups and multiple analysis of variance was used for the repeated measurements. Differences between discrete variables were compared by using univariate chi-square analysis. A p value <0.05 was considered significant.

RESULTS

Patient population: Of the 148 patients included, 9 refused to have a final angiogram and 5 patients, in whom inadequate blood samples were drawn at baseline, were excluded from the final analysis. Thus, 134 patients whose PTCA was performed in 157 vessels constituted the study population.

Baseline biochemical and angiographic determinants of restenosis: Restenosis was detected angio-

TABLE III Selected Clinical Biochemical and Angiographic Variables at Follow Up

Variable	No Restenosis (n = 95)	Restenosis (n = 39)	p Value
Number of lesions	112	45	—
Follow-up (weeks)	24 ± 5	20 ± 8	0.005
LVEF (%)	53 ± 8	54 ± 7	NS
% segmental stenosis	27 ± 12	69 ± 17	0.001
Mean % change from baseline of			
Triglycerides	−2.3 ± 44	0.5 ± 36	NS
Cholesterol	14 ± 22	11 ± 22	NS
HDL cholesterol	18 ± 26	24 ± 24	NS
LDL cholesterol	23 ± 49	17 ± 27	NS

LVEF = left ventricular ejection fraction; other abbreviations as in Table I.

graphically in 39 patients (29%; 45 vessels). Selected demographic, laboratory, and angiographic characteristics of patients with and without restenosis are listed in Tables I and II. Both groups were comparable, and at multivariate analysis no variable was predictive of stenosis; the tendency of a vessel to restenose was not patient-dependent, but rather lesion-related.

Thirty-one of 39 patients (79%; 36 diseased vessels) with restenosis had an HDL cholesterol level ≤40 mg/dl, whereas 62 of 95 patients (64%; 74 vessels) without restenosis had an HDL cholesterol ≤40 mg/dl ($p = 0.095$). Restenosis developed in 31 of 102 vessels (30%) in patients with HDL cholesterol ≤40 mg/dl compared with 10 of 55 vessels (19%) in patients with HDL cholesterol >40 mg/dl ($p = 0.092$). No significant differences were observed when restenosis rates in patients with total cholesterol levels >250 mg/dl (5 patients had cholesterol levels >300 mg/dl) were compared with those in patients with cholesterol levels <250 mg/dl. The same applies for LDL cholesterol levels when patients with LDL cholesterol >160 mg/dl were compared with those with LDL cholesterol <160 mg/dl. This analysis did not take into account whether patients were on a lipid-lowering regimen.

Lipids at follow-up and angiographic restenosis: Time to repeated catheterization was significantly shorter for patients with (20 ± 8 weeks) than without (24 ± 5 weeks) restenosis; $p = 0.005$. Table III presents the percent change from baseline to time of repeated angiography of the various lipid variables examined. In 117 patients (with 132 vessels), complete serial blood specimens were obtained until the concluding angiography at 6 months (27 patients underwent cardiac catheterization earlier because of recurrence of symptoms). The lipid results of these patients are listed in Table IV. Both groups (with and without restenosis) had similar findings, and triglycerides decreased equally in both. During follow-up, apart from total cholesterol, which increased only mildly in the group with restenosis, all other variables increased significantly in each group over time, but no difference was observed between groups with regard to extent of change. No correlation was found between the tendency to restenose and change in plasma lipids over time.

DISCUSSION

This study shows that short-term (6 months) restenosis after PTCA is not determined by plasma levels of total cholesterol, or HDL and LDL cholesterol levels, or by triglycerides at the time of the procedure, if these lipids are not profoundly altered. However, a trend toward increased restenosis rate was observed in vessels of patients who had an HDL cholesterol level ≤40 mg/dl at the time of PTCA (30% restenosis) compared with those who had an HDL cholesterol >40 mg/dl (19% restenosis), $p = 0.092$. During follow-up, the significant increase observed in HDL cholesterol (Table IV) did not affect the final outcome. The tendency to restenose had no correlation with any change in the plasma lipids concentration.

Although the association of serum lipid levels with risk of atherosclerosis is well established, studies that have examined the potential role of serum lipids in the coronary restenosis process have been inconclusive.[2–4] Although some studies have suggested that patients with hypercholesterolemia or low levels of HDL cholesterol are at increased risk for restenosis,[11,12] our results are in agreement with other reports that have been unable to make a convincing association between lipid levels and restenosis.[17,18] Nevertheless, the trend observed in our study for increased restenosis in patients with an HDL cholesterol ≤40 mg/dl corroborates previous observations. Shah and Amin[19] demonstrated a highly signifi-

TABLE IV Plasma Lipids at Baseline and During Follow-Up in 117 Patients Who Underwent Successful Coronary Angioplasty

Variables	Baseline	Months 1	2	3	6	p Value
No restenosis						
Triglycerides	180 ± 74	170 ± 81	159 ± 67	163 ± 90	159 ± 94	NS
Cholesterol	207 ± 42	220 ± 31	220 ± 42	225 ± 35	226 ± 34	0.004
HDL cholesterol	39 ± 8	43 ± 9	45 ± 10	45 ± 11	46 ± 10	0.001
LDL cholesterol	133 ± 30	147 ± 30	149 ± 33	153 ± 32	155 ± 32	0.001
Restenosis						
Triglycerides	170 ± 64	149 ± 49	170 ± 57	145 ± 42	158 ± 58	NS
Cholesterol	202 ± 42	211 ± 30	223 ± 39	223 ± 35	218 ± 42	0.08
HDL cholesterol	37 ± 9	40 ± 8	42 ± 9	45 ± 8	44 ± 9	0.001
LDL cholesterol	132 ± 30	141 ± 26	149 ± 30	153 ± 28	149 ± 34	0.04

Abbreviations as in Table I.

cant relation between a low HDL cholesterol level and restenosis rate after coronary angioplasty. An HDL cholesterol level <40 mg/dl was associated with a nearly fourfold higher restenosis rate than HDL cholesterol levels >40 mg/dl, and a shorter time to restenosis. The level of LDL cholesterol was not significantly related to restenosis.

Our study population did not include patients with extreme serum lipid abnormalities and only a small fraction of patients had total cholesterol levels >300 mg/dl, HDL cholesterol <30 mg/dl, or LDL cholesterol >180 mg/dl. Nevertheless, it has been suggested by Roberts[20] that all patients with coronary artery disease have cholesterol levels that are too high for their vascular biology. The hypothesis that lipid lowering may play a role in restoration of normal endothelial function after PTCA encouraged studies aimed to substantially manipulate lipid metabolism. However, although a beneficial effect of omega-3 fatty acids was reported by some investigators,[21-23] others showed that it had no effect on the incidence of restenosis.[10,24] The same applies to lovastatin in which Sahni et al[25] showed a significant reduction in restenosis rate, while Hollman et al[26] found no advantage using an aggressive lipid-lowering regimen (including lovastatin).

Most studies are limited by the lack of arteriography in asymptomatic patients and lack of lipid fractions or serial lipid measurements during follow-up. The strength of the present study is that all patients underwent coronary angiography, and therefore, patients with silent restenosis were not overlooked. Moreover, serial measurements of blood lipids were obtained (not only 1 baseline measurement).

During follow-up, triglyceride levels decreased compared with those at baseline, whereas plasma cholesterol fractions gradually increased in both patients with and without restenosis (Table IV). The gradual increase in HDL cholesterol is probably a result of increased awareness of patients (after PTCA) to perform physical activity and restrict fat consumption. This may partly explain the increase in total cholesterol although the underlying reason for the increase in LDL cholesterol remains unclear. Interestingly, these changes did not affect the tendency for vessels to restenose.

Our follow-up findings are similar to those of Arora et al,[27] who found that cholesterol levels at the time of PTCA did not predict restenosis, whereas follow-up cholesterol levels showed an inverse relation, contrary to what was expected.

Finally, similar to conclusions obtained from case control and prospective studies,[28] this study did not find any relation between plasma triglyceride levels at baseline or during follow-up and the rate of restenosis. Thus, although lipid levels at the time of PTCA and at 6 months after the procedure were not found to predict the occurrence of restenosis, the association of low high-density lipoprotein levels and the tendency for restenosis to occur should not be overlooked.

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APPENDIX

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